

AMENDMENTS TO THE CLAIMS

This listing of claims will replace all prior versions of claims in the application.

Listing of Claims:

1. (Currently Amended) A method for increasing or maintaining the number of functional pancreatic islet cells ~~of a predetermined type in an organ or tissue a pancreas~~ of a ~~mammal~~ human, wherein said ~~organ or tissue pancreas~~ is injured, damaged, or deficient in said functional pancreatic islet cells, said method comprising administering to said ~~mammal~~ human a composition enriched in pluripotent cells that express the Hox11 gene.
2. (Original) The method of claim 1, further comprising stimulating said organ or tissue before administering said composition.
3. (Original) The method of claim 2, wherein said organ or tissue is stimulated by administering TNF-alpha.
4. (Original) The method of claim 2, wherein said organ or tissue is stimulated by administering a TNF-alpha agonist or a TNF-alpha inducing substance.
5. (Previously Presented) The method of claim 4, wherein said TNF-alpha agonist or TNF-alpha inducing substance is Complete Freund's Adjuvant (CFA), ISS-ODN, microbial cell wall components with LPS-like activity, cholera particles, *E. coli* heat labile enterotoxin, *E. coli* heat labile enterotoxin complexed with lecithin vesicles, ISCOMS-immune stimulating complexes, polyethylene glycol, poly(N-2-(hydroxypropyl)methacrylamide), synthetic oligonucleotides containing CpG or CpA motifs, monophosphoryl lipid A, *Bacillus Clamette-Guerin*, γ -interferon, Tissue Plasminogen Activator, LPS, Interleukin-1, Interleukin-2, UV light, a lymphotoxin, cachectin, a TNFR-2 agonist, an intracellular mediator of the TNF-alpha signaling pathway, a NF κ B inducing substance, IRF-1, STAT1, a lymphokine, a tumor necrosis

factor-alpha (TNF- α) receptor II agonist, or the combination of TNF-alpha and an anti-TNFR-1 antibody.

6. (Original) The method of claim 5, wherein said TNF-alpha agonist or TNF-alpha inducing substance is Complete Freund's Adjuvant, Bacillus Clamette-Guerin, or γ -interferon.

7. (Original) The method of claim 2, wherein said organ or tissue is stimulated 6-12 hours before administering said composition.

8. (Original) The method of claim 1, wherein said composition is enriched in cells which do not express CD45 protein.

9. (Currently Amended) The method of claim 8, wherein said pluripotent cells are enriched from the peripheral blood or tissue of a ~~mammal~~ human by a method comprising: a) providing from the ~~mammal~~ human peripheral blood or tissue that contains pluripotent cells; b) separating pluripotent cells from said peripheral blood or tissue; c) separating said pluripotent cells into a first cell population which expresses CD45 antigen on the surface of said cells and a second cell population which predominantly does not express CD45 antigen on the surface of said cells; and d) selecting said second cell population.

10. (Original) The method of claim 1, wherein said pluripotent cells are derived from the spleen.

11. (Original) The method of claim 1, wherein said pluripotent cells are semi-allogeneic.

12. (Original) The method of claim 1, wherein said pluripotent cells are isogeneic.

13-29 (Canceled)

30. (Currently Amended) The method of claim 1, wherein said composition comprises cells that present MHC class I and peptide, wherein said MHC class I has at least one allele that matches an MHC class I allele expressed by said mammal human.

31-52 (Canceled)

53. (Currently Amended) The method of claim 1, further comprising administering to said mammal human an agent that selectively inhibits, removes, or kills cell populations that interfere or prevent the trafficking of, differentiation of, or growth of Hox-11-expressing pluripotent cells.

54. (Previously Presented) The method of claim 53, wherein said cell populations comprise lymphocytes.

55. (Original) The method of claim 53, wherein said agent comprises TNF-alpha.

56. (Original) The method of claim 53, wherein said agent comprises a TNF-alpha agonist or a TNF-alpha inducing substance.

57. (Previously Presented) The method of claim 56, wherein said TNF-alpha agonist or TNF-alpha inducing substance is Complete Freund's Adjuvant (CFA), ISS-ODN, microbial cell wall components with LPS-like activity, cholera particles, *E. coli* heat labile enterotoxin, *E. coli* heat labile enterotoxin complexed with lecithin vesicles, ISCOMS-immune stimulating complexes, polyethylene glycol, poly(N-2-(hydroxypropyl)methacrylamide), synthetic oligonucleotides containing CpG or CpA motifs, monophosphoryl lipid A, Bacillus Clamette-Guerin, γ -interferon, Tissue Plasminogen Activator, LPS, Interleukin-1, Interleukin-2, UV light, a lymphotoxin, cachectin, a TNFR-2 agonist, an intracellular mediator of the TNF-alpha signaling pathway, a NF κ B inducing substance, IRF-1, STAT1, a lymphokine, a tumor necrosis factor-alpha (TNF- α) receptor II agonist, or the combination of TNF-alpha and an anti-TNFR-1

antibody.

58. (Withdrawn) The method of claim 57, wherein said TNF-alpha agonist or TNF-alpha inducing substance is Complete Freund's Adjuvant, Bacillus Clamette-Guerin, or γ -interferon.

59. (Currently Amended) The method of claim 1, wherein said mammal human has an autoimmune disease.

60. (Original) The method of claim 59, wherein said disease is diabetes.

61. (Withdrawn) The method of claim 59, wherein said disease is immunologically-mediated glomerulonephritis.

62. (Withdrawn) The method of claim 59, wherein said disease is chronic hepatitis, primary biliary cirrhosis, or primary sclerosing cholangitis.